

Amendment to the Claims

Claims 1-33 (Canceled)

34. (Currently amended) ~~The A transgenic mouse of claim 43 whose genome comprises a disruption in the endogenous mouse anaphylatoxin C3a receptor gene, wherein where the mouse disruption is homozygous for said null allele and the mouse is male, the transgenic mouse lacks production of functional anaphylatoxin C3a receptor and exhibits, relative to a wild-type mouse, reduced thymus weight, reduced thymus size, reduced thymus to body weight ratio, increased susceptibility to seizure or a stimulus processing deficit, and wherein where the mouse disruption is homozygous for said null allele and the mouse is female, the transgenic mouse lacks production of functional anaphylatoxin C3a receptor and exhibits, relative to a wild-type mouse, increased susceptibility to seizure or a stimulus processing deficit.~~

35. (Previously presented) The transgenic mouse of claim 34, wherein the increased susceptibility to seizure is characterized by a lower dose of metrazol required to reach characteristic stages of seizure, relative to a wild-type mouse.

36. (Previously presented) The transgenic mouse of claim 34, wherein the stimulus processing deficit is characterized by a decrease in prepulse inhibition, relative to a wild-type mouse.

37. (Previously presented) A cell or tissue obtained from the transgenic mouse of claim 34.

38. (Canceled)

39. (Canceled)

40. (Canceled)

41. (Currently amended) A method of producing a transgenic mouse whose genome comprises a disruption in the endogenous mouse anaphylatoxin C3a receptor gene, the method comprising:

- (a) introducing a targeting construct capable of disrupting the endogenous mouse anaphylatoxin C3a receptor gene into a mouse embryonic stem cell;
- (b) introducing the mouse embryonic stem cell into a blastocyst;
- (c) implanting the blastocyst into a pseudopregnant mouse, wherein the pseudopregnant mouse gives birth to a chimeric mouse; and
- (d) breeding the chimeric mouse to produce the transgenic mouse whose genome comprises the disruption in the endogenous mouse anaphylatoxin C3a receptor gene;

wherein where the disruption is homozygous and the mouse is male, the transgenic mouse lacks production of functional anaphylatoxin C3a receptor and exhibits, relative to a wild-type mouse, reduced thymus weight, reduced thymus size, reduced thymus to body weight ratio, increased susceptibility to seizure or a stimulus processing deficit, and wherein where the disruption is homozygous and the mouse is female, the transgenic mouse lacks production of functional anaphylatoxin C3a receptor and exhibits, relative to a wild-type mouse, increased susceptibility to seizure or a stimulus processing deficit.

42. (Previously presented) The transgenic mouse produced by the method of claim 41.
43. (New) A transgenic mouse whose genome comprises a null endogenous anaphylatoxin C3a receptor allele, said null allele comprising exogenous DNA; said exogenous DNA comprising a gene encoding a visible marker.
44. (New) The transgenic mouse of claim 43 wherein said mouse is heterozygous for said null allele.
45. (New) The transgenic mouse of claim 43 wherein said mouse is homozygous for said null allele.
46. (New) The transgenic mouse of claim 43 wherein said exogenous DNA comprises a gene encoding a selection marker.
47. (New) The transgenic mouse of claim 46 wherein said gene is a neomycin resistant gene.
48. (New) The transgenic mouse of claim 43 wherein said visible marker is lacZ.